PART 12

NEUROLOGIC EXAMINATION

CHAPTER 58

Visual Field Testing

KEY TEACHING POINTS

- Bedside examination is more accurate in detecting posterior visual field defects (produced by lesions in the optic chiasm, optic tracts, optic radiations, or occipital cortex) than anterior defects (lesions in retina or optic nerves).
- Anterior defects affect the patient's visual acuity and pupils; posterior defects do not affect visual acuity and spare the pupils.
- In patients with homonymous hemianopias (suggesting a contralateral post-chiasmal lesion), associated hemiparesis or asymmetric optokinetic nystagmus indicates a parietal lobe lesion; absence of these findings indicates an occipital lobe lesion.
- Detection of visual field defects at the bedside may be improved by special techniques, such as asking the patient to describe the clinician's face (are any features missing?), use of moving red targets, or use of laser pointer projected against a wall.

I. INTRODUCTION

Abnormalities of peripheral vision are called **visual field defects**. These defects, many of which can be detected at the bedside, provide important clues to the diagnosis of lesions throughout the visual pathways—retina, optic nerve, optic chiasm, optic tracts, optic radiations (parietal and temporal lobes), and occipital cortex (Fig. 58.1).

II. DEFINITION

The term **hemianopia** describes visual defects that occupy approximately one-half of an eye's visual space. **Quadrantanopia** describes defects confined mostly to approximately one-fourth of an eye's visual space. **Homonymous** describes defects that affect the same side of the vertical meridian (i.e., right or left side) of both eyes.

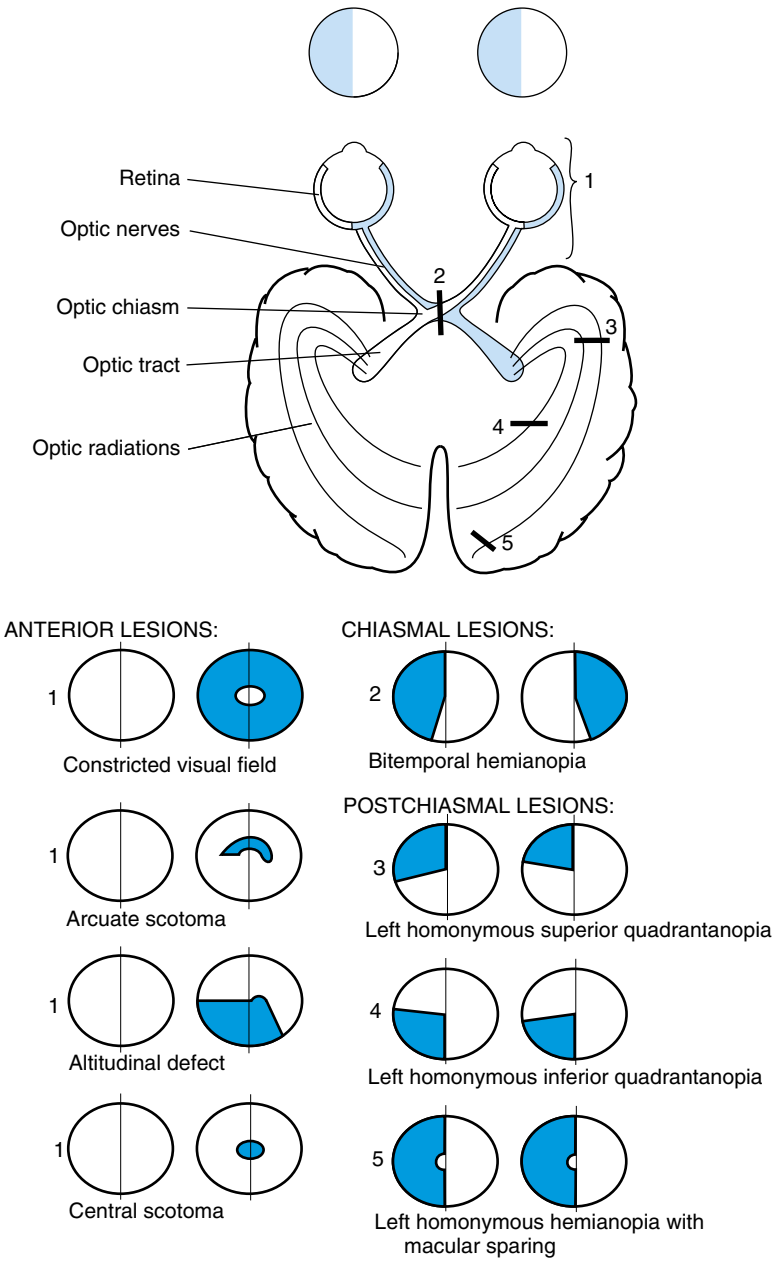

FIG. 58.1 ANATOMY OF THE VISUAL PATHWAYS. The anatomy of the visual pathways appears at the top of the figure, the light blue shading indicating how visual information from the left visual space eventually courses to the right brain. Visual field defects are at the bottom of the figure. Anterior defects (labeled "1," from disease of the optic nerve or retina) characteristically affect one eye and cause defects (the dark blue shading) that may cross the vertical meridian (i.e., the vertical meridian is the vertical line bisecting each visual field). Chiasmal defects (labeled "2") and postchiasmal defects (labeled "3" for a lesion in the anterior temporal lobe, "4" for the parietal lobe, and "5" for the occipital cortex) characteristically affect both eyes and respect the vertical meridian.

For example, a right homonymous hemianopia affects the right visual space of both eyes (i.e., the temporal field of the right eye and the nasal field of the left eye). The term *homonymous* implies the defect does not cross the vertical meridian.

III. THE ANATOMY OF THE VISUAL PATHWAYS

The key anatomic points in Fig. 58.1 are the following: (1) Images from the visual fields are inverted throughout the retina and all neural pathways. Images from the temporal visual field fall on the nasal retina and those from the nasal field on the temporal retina. Images from the *superior* visual fields are transmitted throughout the *inferior* visual pathways (inferior retina, inferior optic nerve and chiasm, and temporal lobe), and those from the *inferior* visual fields throughout the *superior* visual pathways (superior retina, superior optic nerve and chiasm, parietal lobe). (2) The nasal retinal fibers cross in the optic chiasm; therefore, disease of the optic chiasm causes defects in both temporal visual fields (**bitemporal hemianopia**). (3) The visual pathways posterior to the optic chiasm contain information from the same visual space of each eye: lesions in the *right* postchiasmal pathways cause defects in the *left* visual space of each eye (i.e., temporal field of left eye and nasal field of right eye), and those of the *left* postchiasmal pathways cause defects in the *right* visual space. Such defects, respecting the vertical meridian in each eye, are called *homonymous*. (4) The visual pathways in the occipital cortex that contain information from the macula (point of fixation) are distant from those connected to the more peripheral fields.[1] Therefore, lesions of the occipital cortex may cause either homonymous defects sparing the macula or visual defects confined to central vision.

IV. TECHNIQUE

There are many ways to test visual fields at the bedside,[2] but the two traditional techniques are static confrontational testing and kinetic confrontational testing. In all techniques the patient sits approximately 70 to 100 cm from the clinician and fixes on the clinician's own eye. Only one eye of the patient is tested at a time; the other is occluded with a card or the patient's hand.

A. STATIC TECHNIQUE

Using this technique, the clinician presents objects at a fixed point in the visual field, usually approximately 20 to 30 degrees from fixation. The clinician presents one, two, or five fingers to each visual quadrant and asks the patient to count the number of fingers. Testing two quadrants simultaneously (either by asking the patient to count total number of fingers or identify which finger is wiggling) has the advantage of detecting some parietal lobe lesions that may allow patients to see an object in the contralateral field if it appears alone, but not if another object is presented simultaneously to the healthy visual field (i.e., visual extinction).

Throughout the examination, the clinician focuses on whether a defect respects the vertical or horizontal meridians of the visual field (see later). Defects crossing the vertical meridian are due to anterior disease (see later), whereas those respecting the vertical meridian are due to chiasmal disease (if the defect is bitemporal) or postchiasmal disease (if it is homonymous).

B. KINETIC TECHNIQUE

In this technique the clinician tests one quadrant at a time by slowly moving an object (e.g., wiggling finger, <5 degrees of oscillation) from an extreme peripheral

field toward fixation, the patient then indicating the moment he or she sees the object. The trajectory of the moving object is an imaginary line bisecting the horizontal and vertical meridians (e.g., 45, 135, 225, and 315 degrees from the vertical meridian), and the direction of movement is from periphery to central fixation.

V. THE FINDINGS

Visual field defects are classified as *prechiasmal* defects (from disease in retina or optic nerves, often called *anterior* defects), *chiasmal* defects, and *postchiasmal* defects (optic tracts, optic radiations, and occipital cortex).

A. ANTERIOR OR PRECHIASMAL DEFECTS

The characteristic features are the following:

1. **One Eye Is Affected** (unless the retinal or optic nerve disorder is bilateral).
2. **Visual Acuity Is Poor.** Most patients have diminished acuity or, if acuity is normal, other signs of anterior disease, such as an afferent pupillary defect (see Chapter 21), red color desaturation, abnormal retina examination, or an abnormal optic disc (drusen, cupping, or atrophy).
3. **The Defects May Cross the Vertical Meridian.** This occurs because retinal nerve fibers from the temporal retina arch across the vertical meridian to reach the optic disc and nerve (which lie on the nasal side of the retina). Damage to these fibers thus may cause a defect that crosses the vertical meridian. Small nerve fiber defects may cause an *arcuate defect* (see Fig. 58.1), larger ones an *altitudinal defect* (having a sharp horizontal border in the nasal field). Damage to fibers from the macula may cause *central scotomata* and, to those preferentially affecting the most peripheral vision, *constricted visual fields*.[3]

B. CHIASMAL DEFECTS

These defects are bitemporal hemianopias (see Fig. 58.1).

C. POSTCHIASMAL DEFECTS

The characteristics of these defects are the following:

1. **Both Eyes Are Affected,** causing homonymous hemianopias or quadrantanopias.
2. **Visual Acuity Is Normal.** This is true in greater than 90% of cases. If visual acuity is abnormal, it is because of bilateral disease and thus the acuity in both eyes is the same.[4]
3. **Pupil and Retinal Examinations Are Normal.** One important exception is the occasional finding of papilledema, caused by brain tumors affecting the optic radiations.

VI. CLINICAL SIGNIFICANCE

A. ETIOLOGY

Most anterior defects are caused by severe glaucoma, retinal emboli, and optic neuritis.[2] Chiasmal defects are usually from a pituitary tumor just below the optic chiasm. More than 95% of postchiasmal defects are due to lesions of the temporal, parietal, and occipital lobes. Lesions of the optic tracts are uncommon.[4,5]

Although parietal and temporal lobe disease may cause inferior and superior quadrantanopias, respectively (see Fig. 58.1), lesions in these areas more often cause dense hemianopias or hemianopias that are denser inferiorly or superiorly, respectively.[4,6]

B. DIAGNOSTIC ACCURACY

EBM Box 58.1 summarizes the diagnostic accuracy of the confrontational technique for diagnosing visual field defects. According to these likelihood ratios (LRs), the finding of a visual field defect by confrontation significantly increases the probability that one is actually present (i.e., by perimetry, LRs = 5.7 to 9.6). Nonetheless, the absence of a defect on bedside testing only modestly decreases the probability of an actual defect (especially for anterior defects, LR = 0.7). Sensitivity is lower for anterior defects because anterior defects are much less dense than posterior ones (see the section on Improving Detection of Visual Defects).[2]

C. DIFFERENTIAL DIAGNOSIS OF POSTCHIASMAL DEFECTS

Homonymous hemianopias may be either an isolated finding or associated with other neurologic findings. The most common cause of an *isolated* homonymous hemianopia is an ischemic infarct of the occipital cortex.[13,14] In patients with associated hemiparesis, aphasia, or asymmetric optokinetic nystagmus, the most common diagnosis is parietal lobe disease.[4,13,15,16] Optokinetic nystagmus is a normal horizontal nystagmus that occurs when patients look at a vertically striped tape moving in front of them. The clinician moves the tape first to one side and then the other, comparing the amplitude of horizontal nystagmus produced, which should be equal in each direction.

EBM BOX 58.1
*Visual Field Defects**

Finding (Reference)†	*Sensitivity (%)*	*Specificity (%)*	*Likelihood Ratio‡ if Finding Is*	
			Present	*Absent*
***Confrontation Technique, Detecting the Following Visual Field Defects*[2,7-12]**				
Anterior defects (retina and optic nerve)	11-58	93-99	**5.7**	0.7
Patchy defects	6			
Constriction of visual fields	58			
Arcuate defects	20-51			
Altitudinal defects	88			
Posterior defects (optic chiasm to occipital cortex)	43-86	86-98	**9.6**	0.4
Bitemporal hemianopia	45			
Homonymous hemianopia	80			
Patients With Homonymous Hemianopias, Detecting Parietal Lobe Disease				
Asymmetric optokinetic nystagmus[4]	93	84	**5.7**	**0.1**
Associated hemiparesis or aphasia[13]	90	95	**18.3**	**0.1**

*Diagnostic standard: for *visual field defects,* conventional perimetry.
†Definition of findings: abnormal static finger counting, static kinetic finger testing, kinetic finger boundary testing, or combinations of these tests.
‡Likelihood ratio (LR) if finding present = positive LR; LR if finding absent = negative LR.
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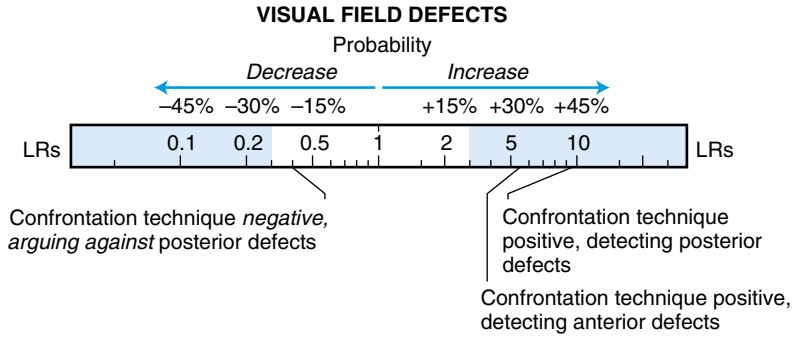

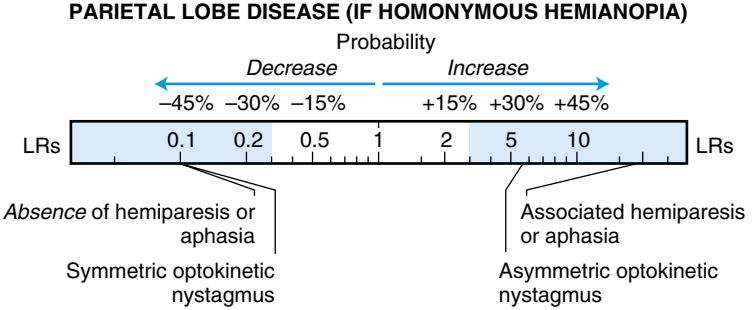

Parietal lobe lesions reduce or eliminate optokinetic nystagmus when the tape is moved *toward* the side with the lesion (Barany first made this observation in 1921).

In patients undergoing computed tomography of the head (because of stroke, headache, seizures), the finding of a homonymous hemianopia increases the probability of contralateral focal cerebral disease (sensitivity 22% to 30%, specificity 93% to 98%, positive LR 4.3; see Chapter 61).[17,18] In those patients with homonymous defects the presence of asymmetric optokinetic nystagmus, associated aphasia or hemiparesis, increases the probability of a parietal lobe lesion (LR = 5.7 for optokinetic nystagmus and LR = 18.3 for hemiparesis or aphasia), whereas the absence of these findings decreases the probability of a parietal lobe lesion (both LRs = 0.1) and thus makes occipital or temporal lobe disease more likely.

D. IMPROVING DETECTION OF VISUAL FIELD DEFECTS

Confrontation fails to detect some defects because they are too small, lack a sharp linear border (e.g., patchy defects of anterior disease), or are too peripheral (e.g., constricted visual fields; confrontation tests only the most central 20 to 30 degrees of visual space). To increase sensitivity of bedside examination, some experts have proposed increasing the distance between clinician and patient during testing from 1 to 4 m, which may improve the detection of subtle arcuate scotomata (glaucoma or optic nerve disease) or macular sparing (some occipital cortex lesions).[19] Additional techniques include: (1) **description of face**: The patient is asked to report if any part of the examiner's face is distorted or missing; (2) **kinetic red boundary testing**: The patient is asked to report when a moving red target (5- to 20-mm diameter) first appears as it is moved inward from the periphery; (3) **red target comparison**: The

examiner presents two 20-mm red targets (often the caps of mydriatic solutions) to two quadrants simultaneously and asks the patient if the bottle tops appear equally red; and (4) **laser target testing**: The clinician uses a conventional red laser pointer and projects it in front of the patient on a screen 1 m away.[10,20]

According to studies comparing these various techniques (EBM Box 58.2), static finger counting, kinetic finger boundary testing, and description of the clinician's face have similar diagnostic accuracy (LR = 13.3 to 54.4). Kinetic testing with a red target and laser pointer testing improve sensitivity but at the cost of diminished specificity. In these studies, the red target comparison test was diagnostically unhelpful (LRs not significant).

EBM BOX 58.2
*Visual Field Testing: Comparison of Techniques**

Finding (Reference)†	*Sensitivity (%)*	*Specificity (%)*	Likelihood Ratio‡ if Finding Is	
			Present	*Absent*
Finger counting[2,11]	25-35	99-100	**54.4**	0.7
Kinetic finger boundary testing[2,11]	39-41	97-99	**13.3**	0.6
Description of face[2,11]	36-44	99	**26.4**	0.6
Kinetic red boundary testing[2,11]	56-74	93-99	**13.6**	0.4
Laser target testing[10]	71	89	**6.3**	**0.3**
Red target comparison[2,11]	59-77	27-99	NS	NS

*Diagnostic standard: for *visual field defect,* conventional perimetry testing (most patients in these studies had anterior visual field defects).
†Definition of findings: for *kinetic red boundary testing,* the moving target was either 5 mm^2 or 20 mm^{11} in diameter and the patient was asked to report when it first appeared red. For all other findings, see the text.
‡Likelihood ratio (LR) if finding present = positive LR; LR if finding absent = negative LR.
NS, Not significant.
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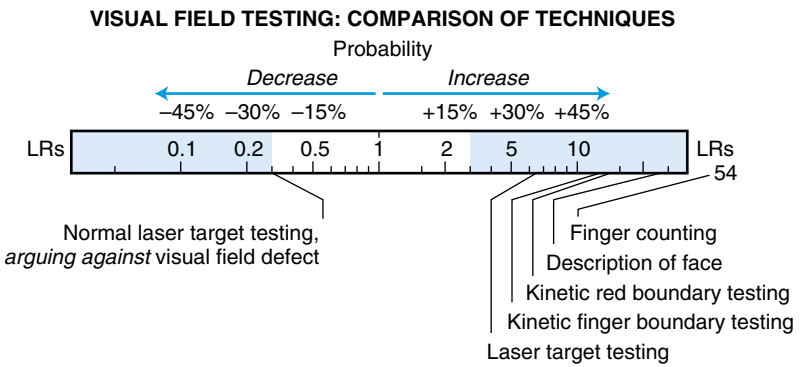

The references for this chapter can be found on www.expertconsult.com.

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